

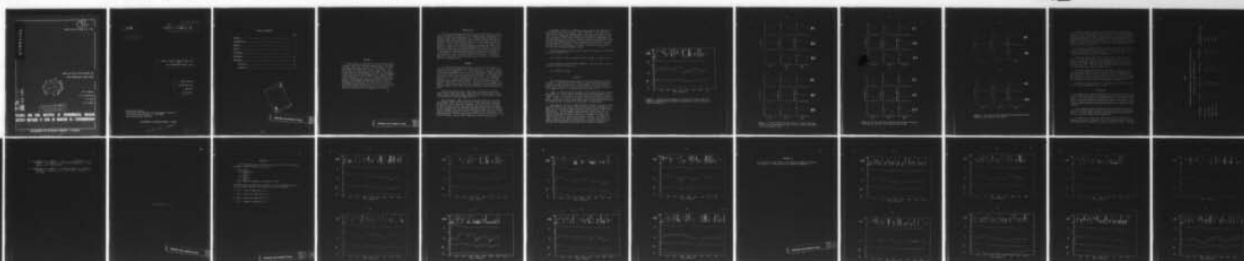
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DCIEM Technical Report No. 77X15

EFFECT OF COLD ON EEG PATTERNS AND  
BODY TEMPERATURE DURING SLEEP



A.G.C. Buguet

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6 EFFECT OF COLD ON EEG PATTERNS AND  
BODY TEMPERATURE DURING SLEEP.

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S.D. / Livingstone,  
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ABSTRACT

→ Sleep stages, as measured by electroencephalographic tracings, and rectal and skin temperatures of two young Caucasian males were recorded for eight nights in test environments at North Bay, Ontario, in February, 1975. During the first three and last two nights, they slept in thermoneutral environments and during the intermediate nights they slept in an unheated tent at ambient temperatures ranging from  $-3^{\circ}\text{C}$  to  $-12^{\circ}\text{C}$ . Both subjects experienced a deprivation of rapid eye movement (REM) sleep; however, no 'rebound' phenomenon was seen on return to thermoneutral conditions. In confirmation of earlier well-substantiated observations, it was observed that the subject who showed more delta sleep in the cold was the one with the greater rectal temperature depression.

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## INTRODUCTION

On a recent military/scientific exercise (Honky Tonk II, Jan-Feb 1975), two men were exposed to cold throughout their designated sleep period for 10 consecutive nights (2). A deprivation of rapid eye movement (REM) sleep relative to control values detected during this period was most pronounced during the first three nights of the cold exposure. On the first recovery night, i.e., the first night during which the subjects slept in thermoneutral conditions after the cold exposure, a REM sleep "rebound" phenomenon was observed. Because the REM deprivation was most pronounced during the initial cold exposure, it was decided to perform an experiment on another pair of subjects to determine if a REM "rebound" phenomenon would occur on return to warm conditions after only three days of cold exposure. This paper describes the results of such an experiment carried out at North Bay, Ontario, in February 1975.

## METHODS

Two young Caucasian male volunteers, aged 22 and 24, served as subjects for the experiment which lasted eight days. The first three nights (ambient temperature,  $T_a = 25^{\circ}\text{C}$ ) were used to establish control or baseline measurements in an electroencephalographic (EEG) laboratory in which the subjects slept in beds under blankets. These nights were followed by three others in which the subjects slept in an unheated tent ( $T_a = -5$  to  $-10^{\circ}\text{C}$ ) using only the outer layer of the two-layer Canadian Forces sleeping bag ensemble, a liner, head hood and an air mattress (insulation = 5.6 clo (10)). During the next two nights, the subjects slept indoors under the same conditions as existed during the baseline nights so that the "recovery" phenomena could be properly examined.

During the baseline and recovery days, the subjects performed normal indoors laboratory work. During the cold exposure days, they stayed outside while performing light exercises in an attempt to duplicate conditions on Exercise Honky Tonk II (2).

Electromyography (EMG), electro-oculography (EOG) and electroencephalography (EEG) were recorded using a Grass Instrument Polygraph, model 8-10 B, with 4 channels assigned to each subject; one channel for EMG recorded from the chin using two electrodes (E4S); one channel for EOG recorded from two electrodes (Beckman 650951); and the remaining two channels were used for EEG which was recorded from 6 electrodes (E5G) in bipolar montage (2 frontal, 2 temporal and 2 occipital). The recommendations of Rechtschaffen and Kales (10) were used to analyze the sleep recordings, and the sleep stages were scored every minute.

Equipment developed at DCIEM was used to measure body temperatures. Seven thermistors were sewn into thermal underwear at the neck (T1), forearm (T2), wrist (T3), abdomen (T4), thigh (T5), calf (T6) and ankle (T7). Rectal temperature was measured by a thermistor inserted 12 cms into the rectum. These eight channels of temperature information from the thermistors were coded into a single electrical signal by using time division multiplexing containing the temperature information in a form of pulse duration modulation. The signal obtained was used to modulate the frequency of a transmitter, the signal of which was detected by a receiver and recorded on magnetic tapes.

Mean skin temperatures ( $\bar{T}_s$ ) were calculated according to the formula of Hardy and DuBois (4):

$$\bar{T}_s = 0.07T_1 + 0.14T_2 + 0.05T_3 + 0.35T_4 + 0.19T_5 + 0.13T_6 + 0.07T_7$$

The calculations of mean body temperature ( $T_b$ ) were made using the weighting factors of Burton (3):

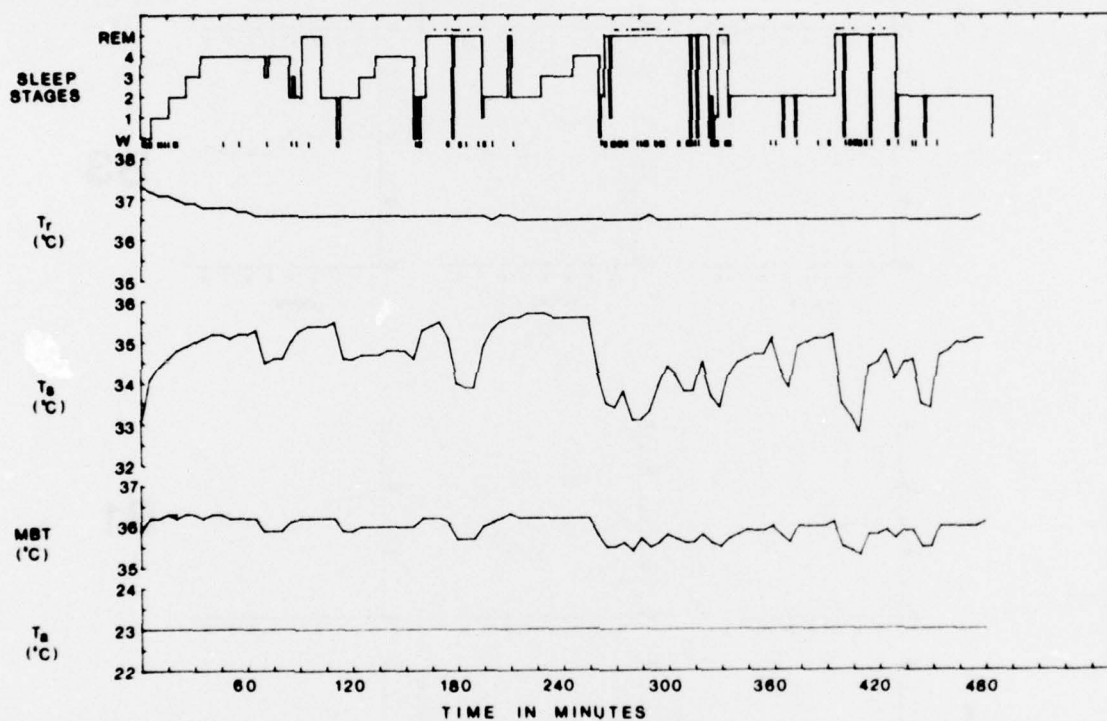
$$T_b = 0.67T_{re} + 0.33\bar{T}_s$$

## RESULTS

The anthropometric characteristics of the two subjects are shown in Table 1. The results of the sleep and temperature studies were summarized in graphs of which Fig. 1 is an example. The first baseline night was not included in the calculation of sleep studies in the baseline summary because of a possible first night effect (1).

Subject 1 (S1) was a good "sleeper" as determined from his baseline hypnograms, i.e. there were no interruptions in the progression in his stages of sleep (9) and 23% of his total sleep time was spent in delta sleep. Subject 2 (S2) was not as good a sleeper since he had less delta sleep, approximately 13% of total sleep time, and less REM sleep than S1. His sleep was also interrupted by more periods of wakefulness.

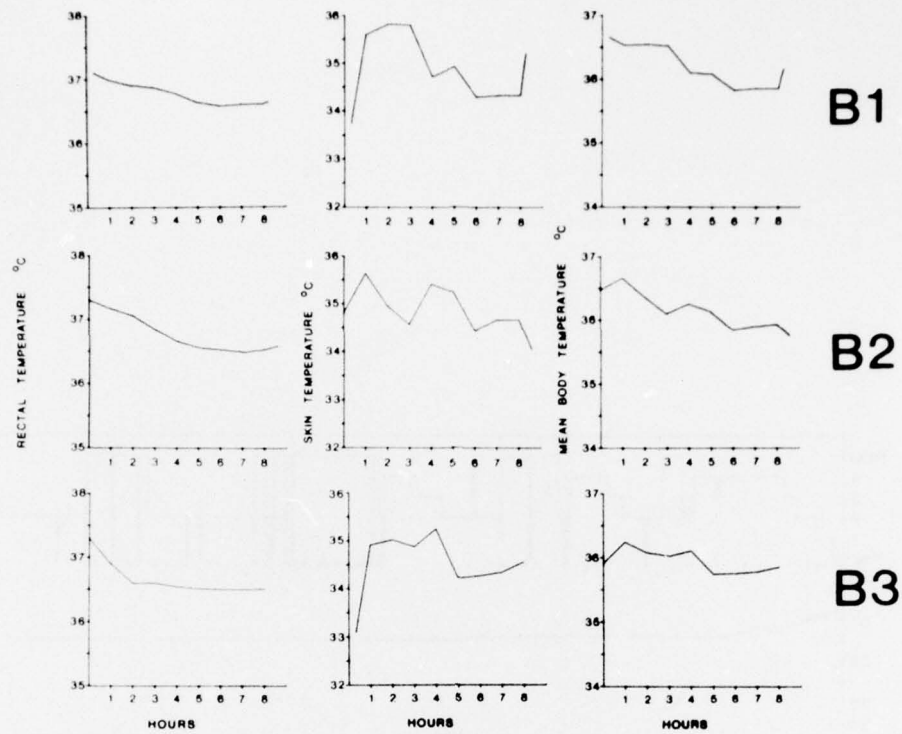
When exposed to cold, S1 showed definite increases in stage 1 sleep and wakefulness of the order of 42% of baseline values, especially on night C1. He also had a slight decrease in stages 3 and 4 (93% of baseline values). He experienced a great decrease in REM sleep (46% of baseline) on C1 with a smaller deprivation on nights C2 and C3. The amount of REM sleep increased on return to thermoneutral conditions (nights R1 and R2) but did not reach baseline values. The amount of stage 2 sleep did not change significantly. The time required to fall asleep increased slightly in the cold but the amount of wakefulness overnight was significantly increased (249% of baseline levels).



**Figure 1:** Composite graph showing the evolution of sleep stages and temperature measurements throughout a representative night (see Appendices for details).



S1



S2

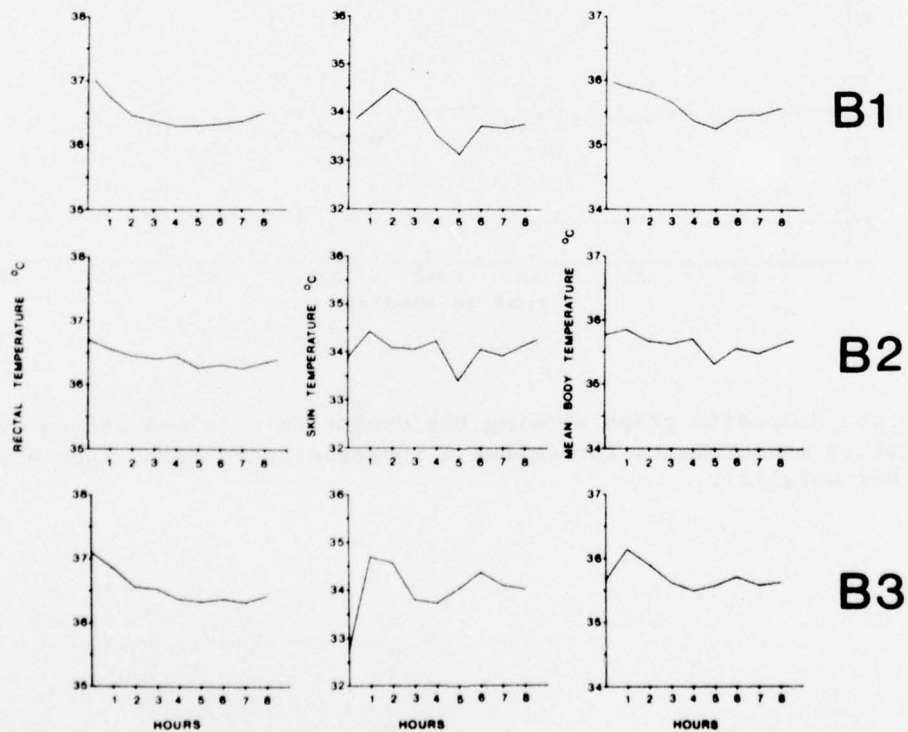
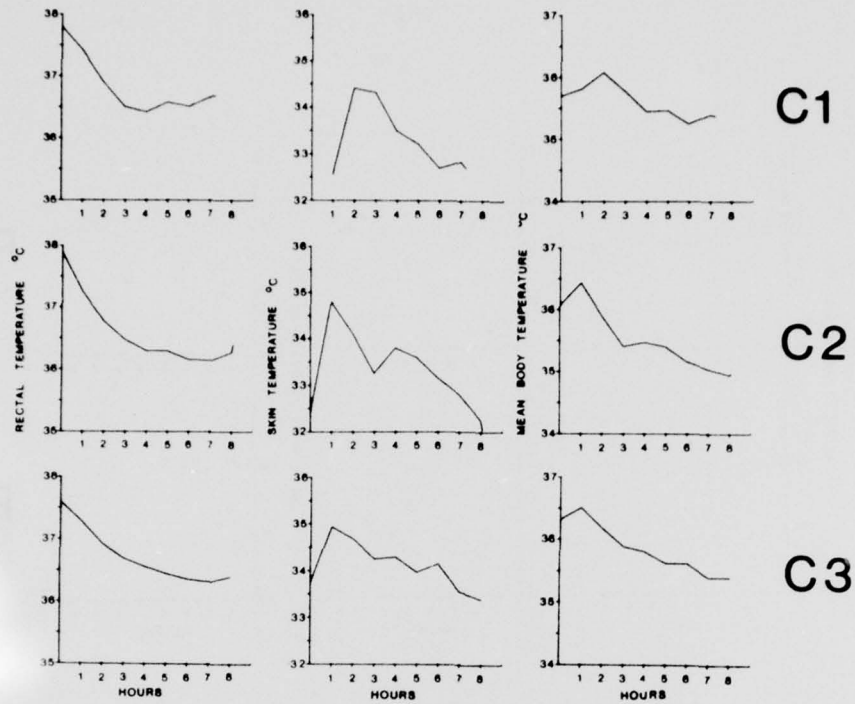


Figure 2: Graphs showing the time course of rectal, mean skin and mean body temperatures during the control nights for subject 1 (S1) and subject 2 (S2).

S1

5



S2

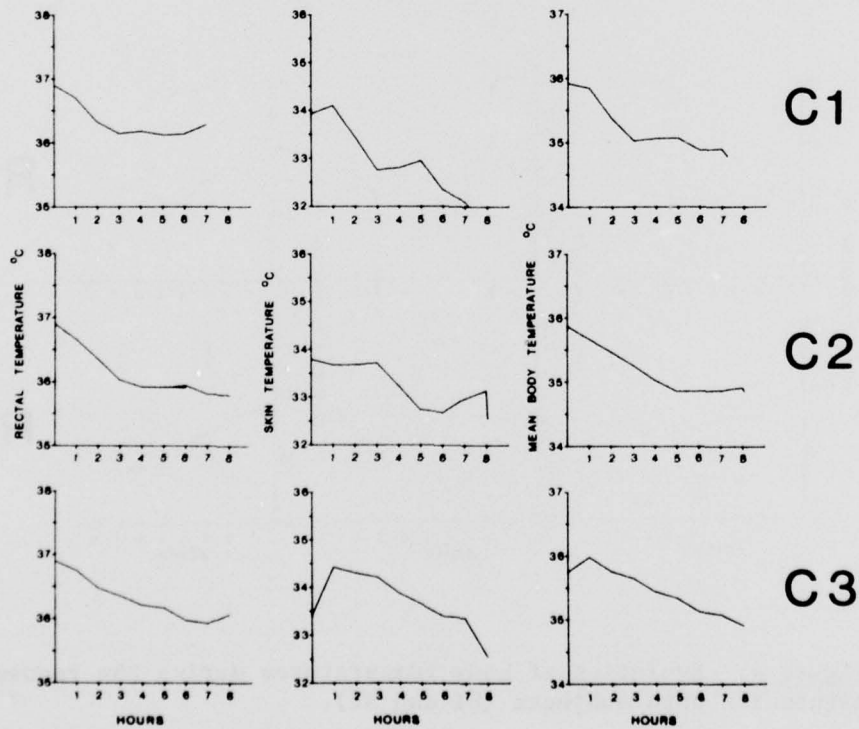
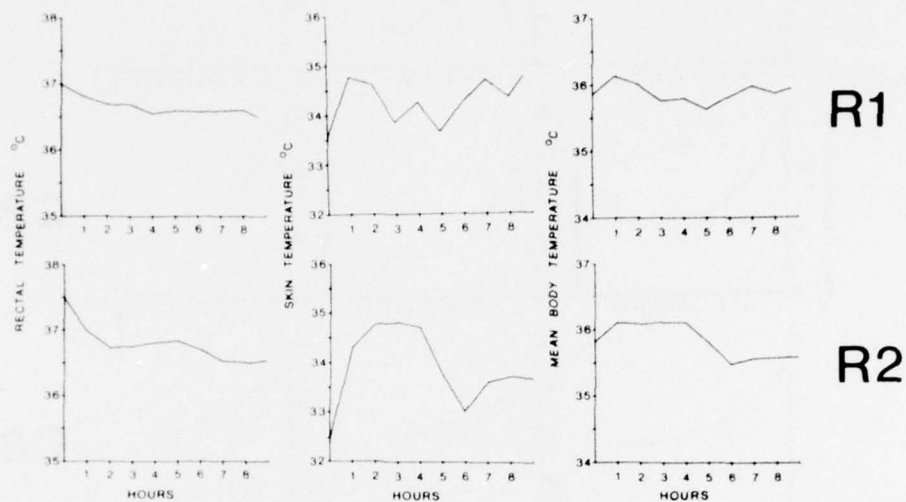


Figure 3: Rectal, mean skin and mean body changes during the cold exposure night for both subjects (S1 and S2).

S1



S2

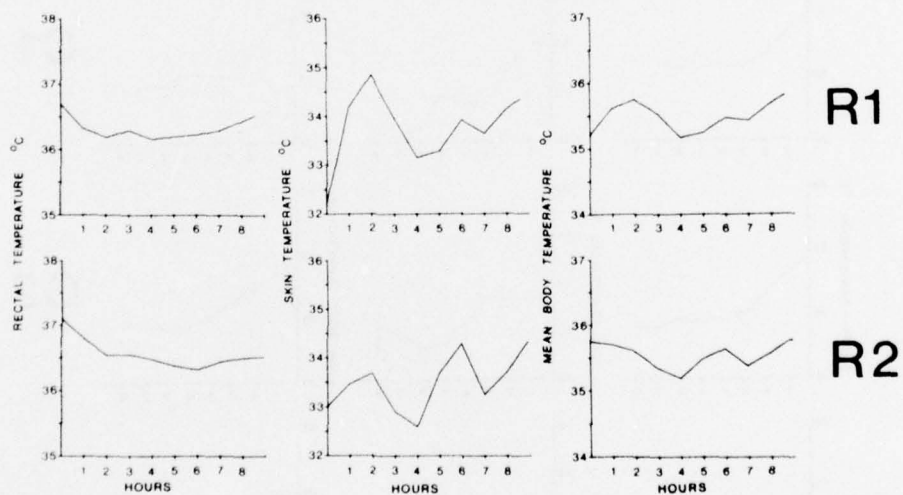


Figure 4: Evolution of body temperatures during the recovery nights for both subjects (S1 and S2).

In the cold, S2, in general, reacted similarly to S1 in that stage 1 sleep increased, stages 3 and 4 initially decreased and REM sleep decreased. Differences showed up in that, for S2, stages 3 and 4 showed an increase on night C3 and there was a progressive decrease in REM sleep from C1 to C3 (reaching 62% of baseline values). REM sleep, in contrast was greatest on C1 for S1. The time to fall asleep did not change in the cold for S2, however overnight wakefulness increased (18% of baseline levels).

Both subjects showed an increase in movement times when sleeping in the cold (147% of baseline values for S1 and 231% for S2). On return to thermoneutral conditions (R1), all sleep parameters showed a tendency to return to baseline conditions.

The rectal temperatures ( $T_{re}$ ) of both subjects fell  $0.5^{\circ}\text{C}$  to  $0.8^{\circ}\text{C}$  until the fourth hour of sleep during the control period and then rose gradually until the subjects awakened. After an initial increase, mean skin temperature ( $T_s$ ) remained between  $33^{\circ}\text{C}$  and  $35^{\circ}\text{C}$  for the whole night. The mean body temperature ( $T_b$ ) fell until the 5th or 6th hour and then rose slowly (Fig. 2).

During the nights involving cold exposure, measurements of  $T_{re}$  in both subjects showed a greater decrease while sleeping and more time transpired before an increase began (6.7 hrs). The drop in  $T_{re}$  was greater in S1 ( $1.5^{\circ}$  to  $2^{\circ}\text{C}$ ) than in S2 ( $0.9^{\circ}$  to  $1.0^{\circ}\text{C}$ ).  $T_s$  dropped constantly after an initial increase but did not fall below  $32^{\circ}\text{C}$ .  $T_b$ , in general, dropped constantly throughout the night (Fig. 3).

During the recovery phase  $T_{re}$ ,  $T_s$  and  $T_b$  varied in much the same way as during the baseline phase. (Fig. 4)

## DISCUSSION

The patterns of sleep seen during cold exposure were similar to those observed during the first three nights of cold exposure on Honky Tonk II. The great amount of wakefulness seen in S1 on night C1 was due to a lack of communication between the subject and observers, the subject being kept awake by a desire to urinate. The amount of stage 1 sleep in both subjects increased generally in the cold as did wakefulness.

It is interesting to note that differences between subjects in the present study resemble subject differences observed during Honky Tonk II. In both cases, the subject who showed more delta sleep in the cold also had a lower rectal temperature.

In Table 1, it can be seen that S1 has a lower surface area to weight ratio than S2. Because of this, it would seem that S2 should cool more than S1; however, the contrary situation occurred. On Honky



TABLE 1

COMPARISON OF ANTHROPOMETRIC CHARACTERISTICS OF THE SUBJECTS AT

NORTH BAY WITH THOSE ON HONKY TONK II

Experiment	Subject	Height (cm)	Weight (Kg)	Surface Area (m <sup>2</sup> )	Surface Area to Weight ratio ( $\frac{m^2}{kg}$ )
North Bay	1	171	76.4	1.89	.0247
North Bay	2	174	65	1.78	.0274
Honky Tonk II	1	168	70.5	1.80	.0255
Honky Tonk II	2	174	86	2.00	.0232

Tonk II, S1, who had the higher surface area to weight ratio did cool more. Thus it seems likely that the amount of cooling is not directly related to body size for the subjects in this experiment.

The effects of cold on sleep are in agreement with those found by Scholander et al (13) when Australian Aboriginies were compared with Caucasians sleeping at night in a cold environment. The Aboriginies slept longer and allowed their body temperatures to drop lower than the Caucasians. In addition Scholander et al (14) found that after cold acclimation in the mountains of Norway, Norweigen subjects slept soundly but kept their body temperatures high. It can be concluded from our results that the subject who has more delta sleep allows himself to cool while the man who has less delta sleep remains warmer. Conversely, the man who cools has more delta sleep than the man who endeavors to keep warm.

No rebound phenomenon of REM sleep was seen on recovery nights in contrast to the finding which occurred after 10 nights in the cold. It may be that the deprivation of REM sleep, although the same for each night, did not accumulate to a sufficient degree over the deprivation period to provide for this rebound.

The lack of rebound might have been caused by the difference in the ambient temperature to which the subjects were exposed. In particular, the faces of these subjects were probably not cooled as much as for the case of subjects on Honky Tonk II because of the higher environmental temperature. If the hypothalamus is cooled by excessive heat loss from the lateral part of the face (3, 6, 7) one could argue that, in the present experiment, the cooling of certain parts of the central nervous system was not as great as Honky Tonk II. Therefore, the deprivation of REM sleep occurring during the three day cold exposure in North Bay may have been an unspecific response to the cold stress. This differs from the Honky Tonk II experiment in that the stress period was carried out for a longer time and that the first unspecific stress response may have been replaced after 4 or 5 days by a specific cold stress response leading to a constant level of REM sleep values that was lower than baseline values. An explanation for the absence of a rebound phenomenon on this experiment may be that the absence of a specific stress was associated with a possible cumulative effect of the REM sleep deprivative.

In conclusion, this experiment has shown that an acute cold exposure provokes a REM deprivation and has confirmed the fact that the subjects who exhibit delta sleep allow their body temperatures to cool.

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## APPENDICES

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## APPENDIX 1

The following figures represent the time course for the following parameters during each night for subject 1

## 1. Sleep stages

w = wakefulness

1 = stage 1

2 = stage 2

3 = stage 3

4 = stage 4

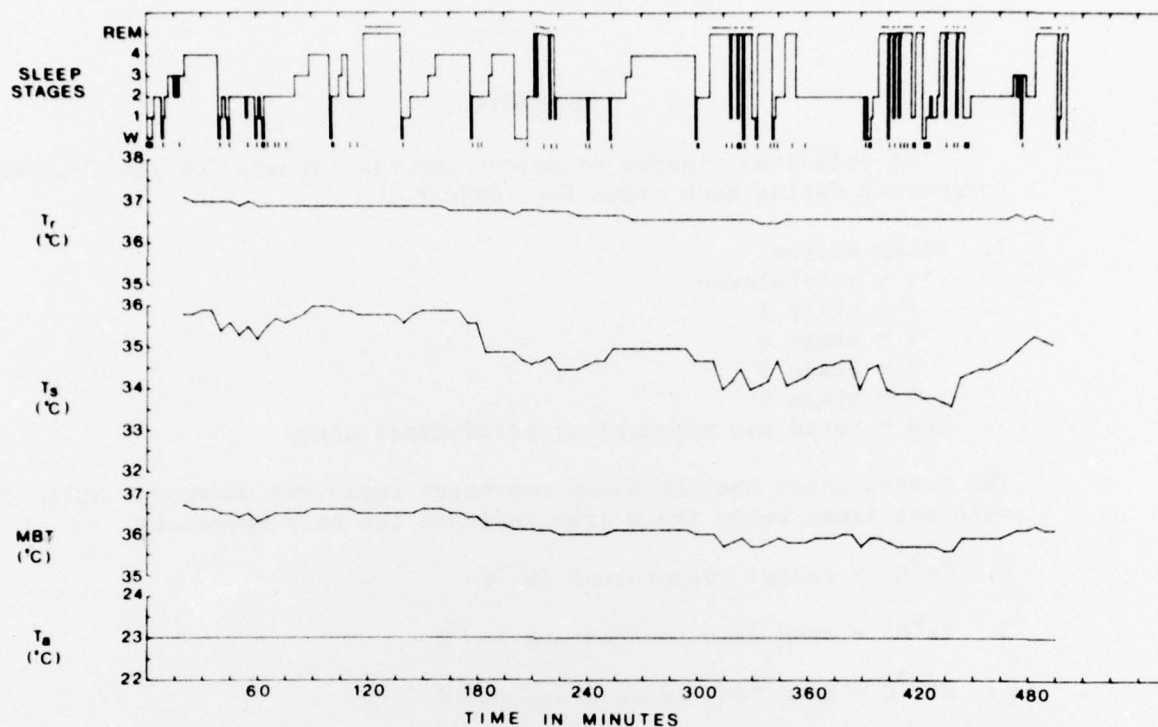
REM = rapid eye movement or paradoxical sleep

The dashes above the REM sleep represent rapid eye movements while the vertical lines below the w line indicate the body movements.

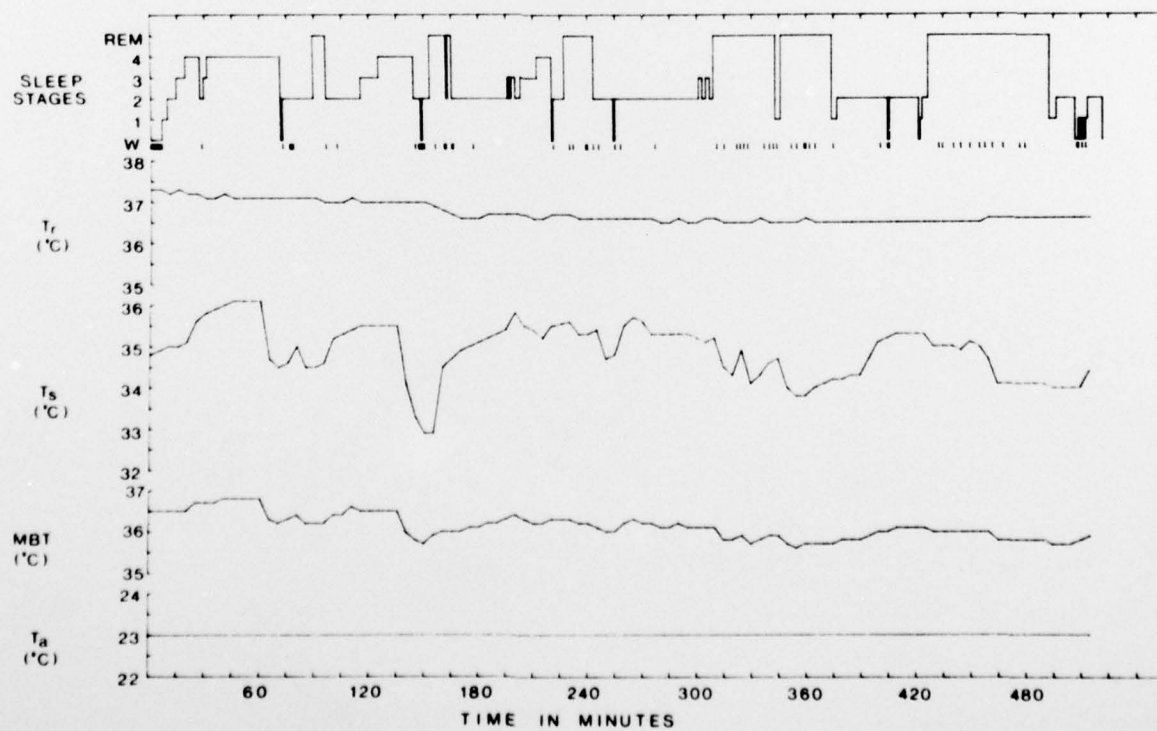
2.  $Tr^{\circ}C$  = rectal temperature in  $^{\circ}C$
3.  $Ts^{\circ}C$  = mean skin temperature in  $^{\circ}C$
4.  $MBT^{\circ}C$  = mean body temperature in  $^{\circ}C$
5.  $Ta^{\circ}C$  = ambient temperature in  $^{\circ}C$

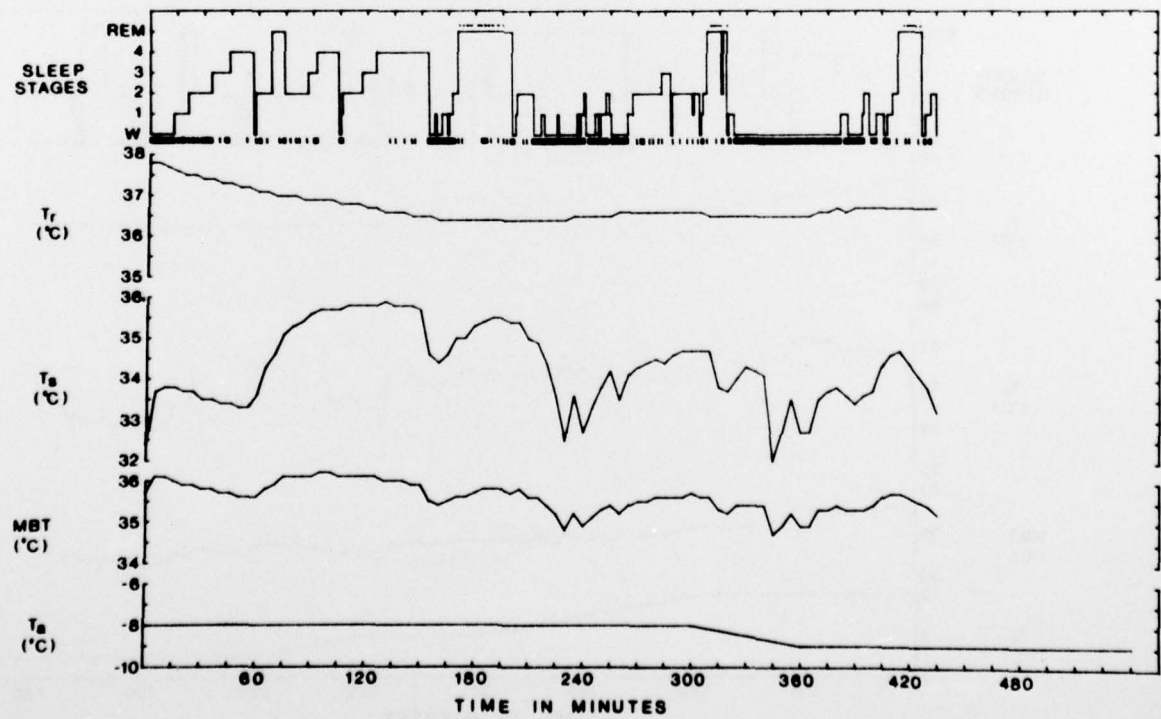
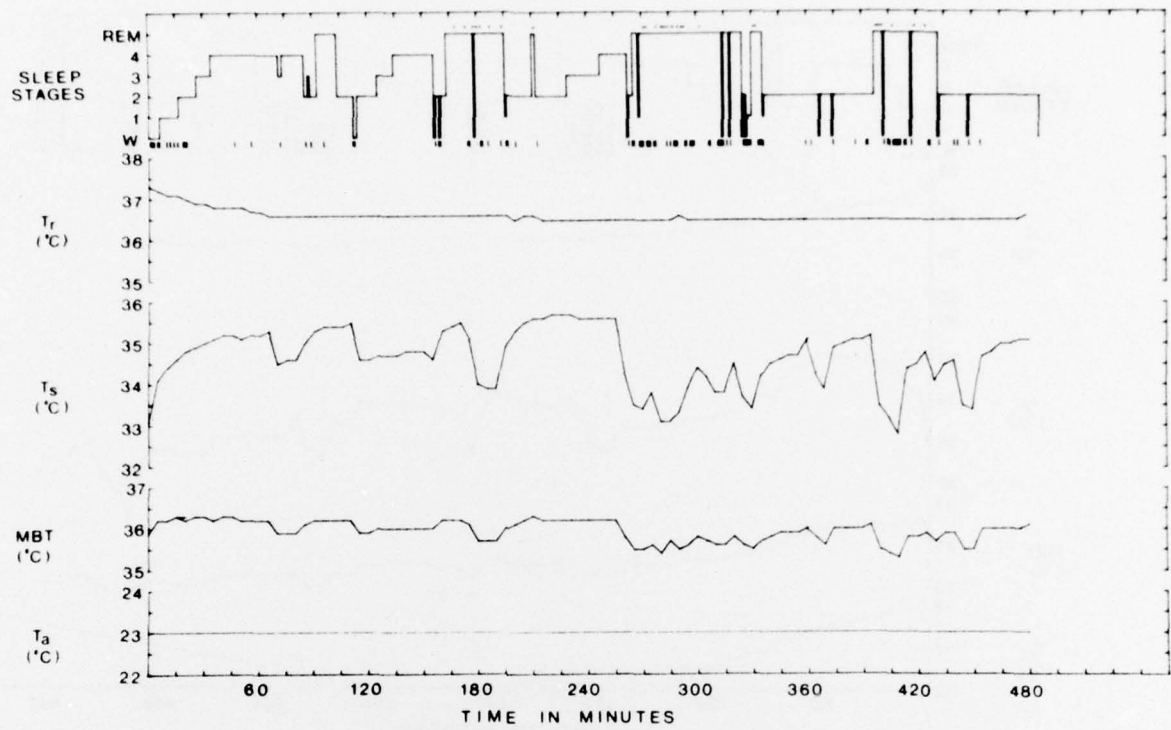
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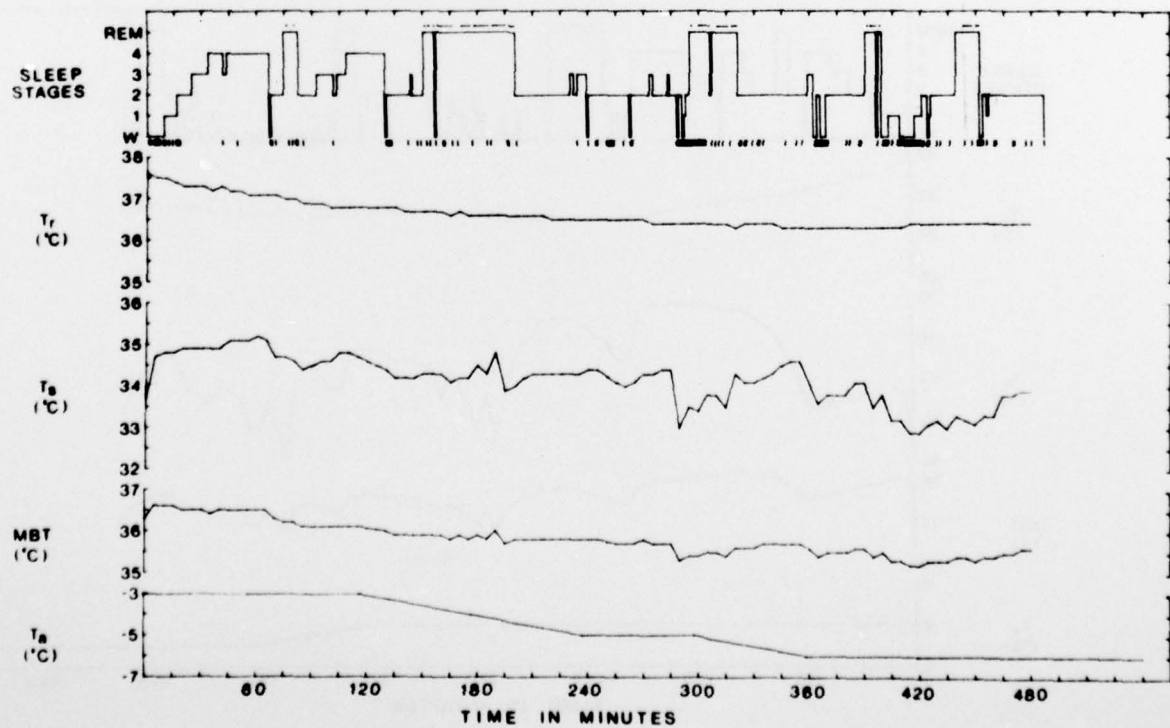
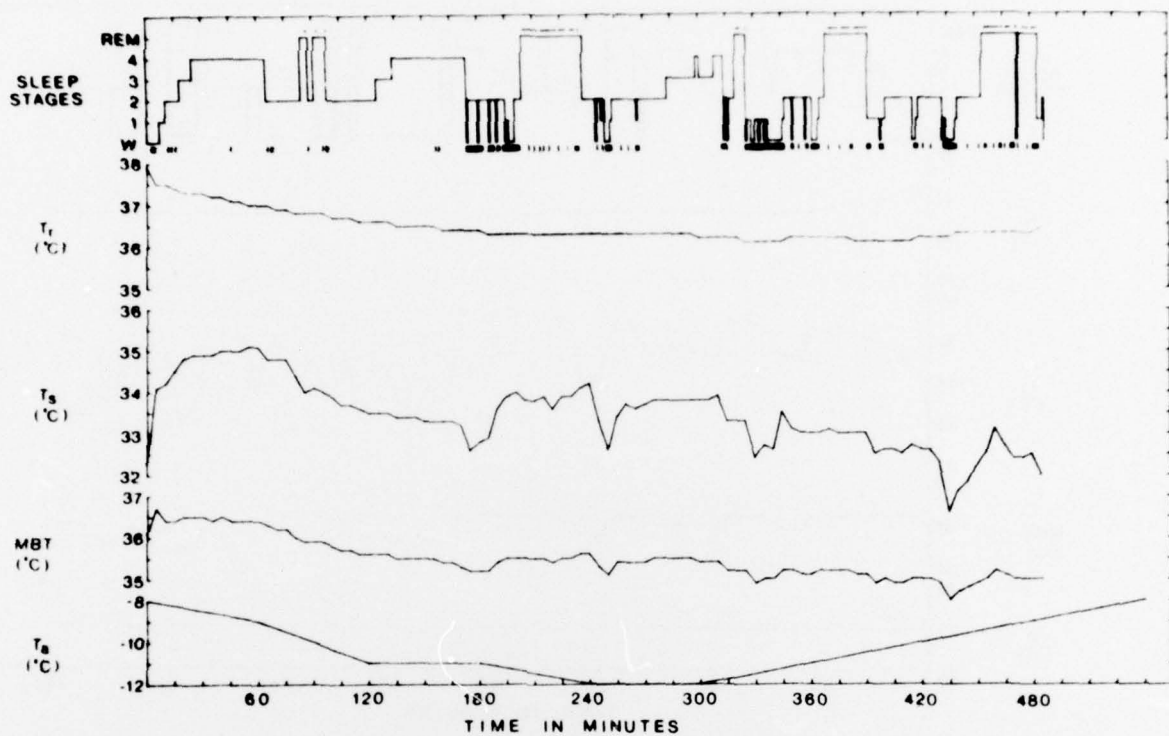


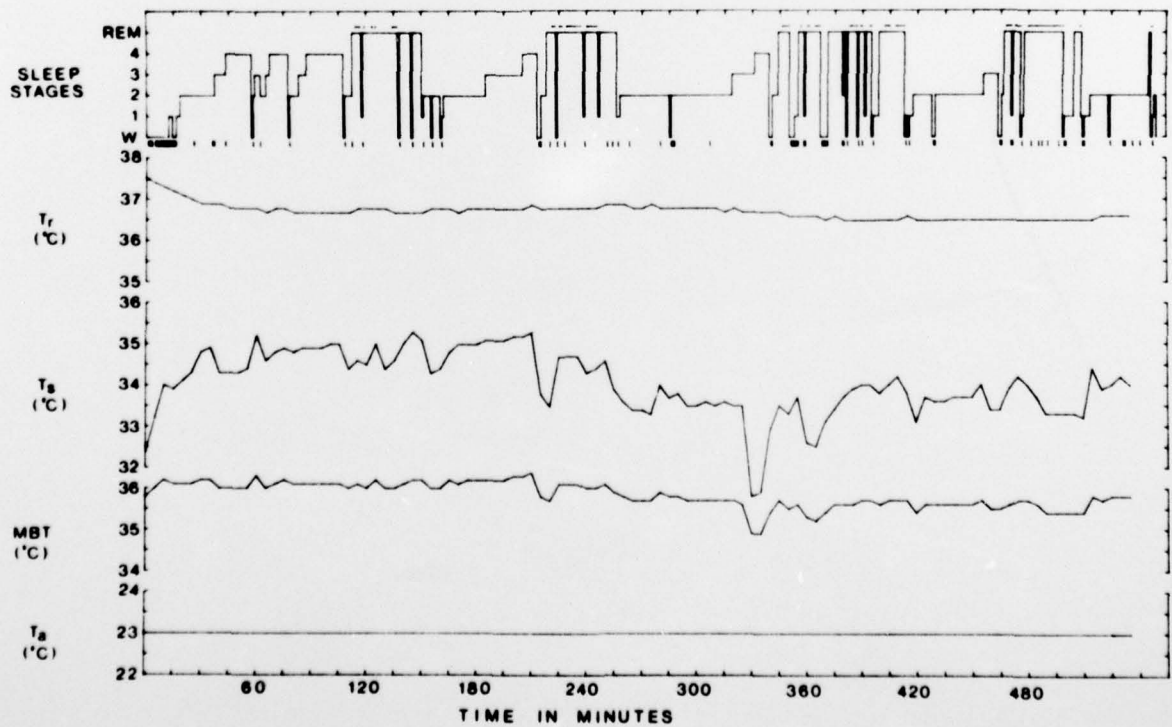
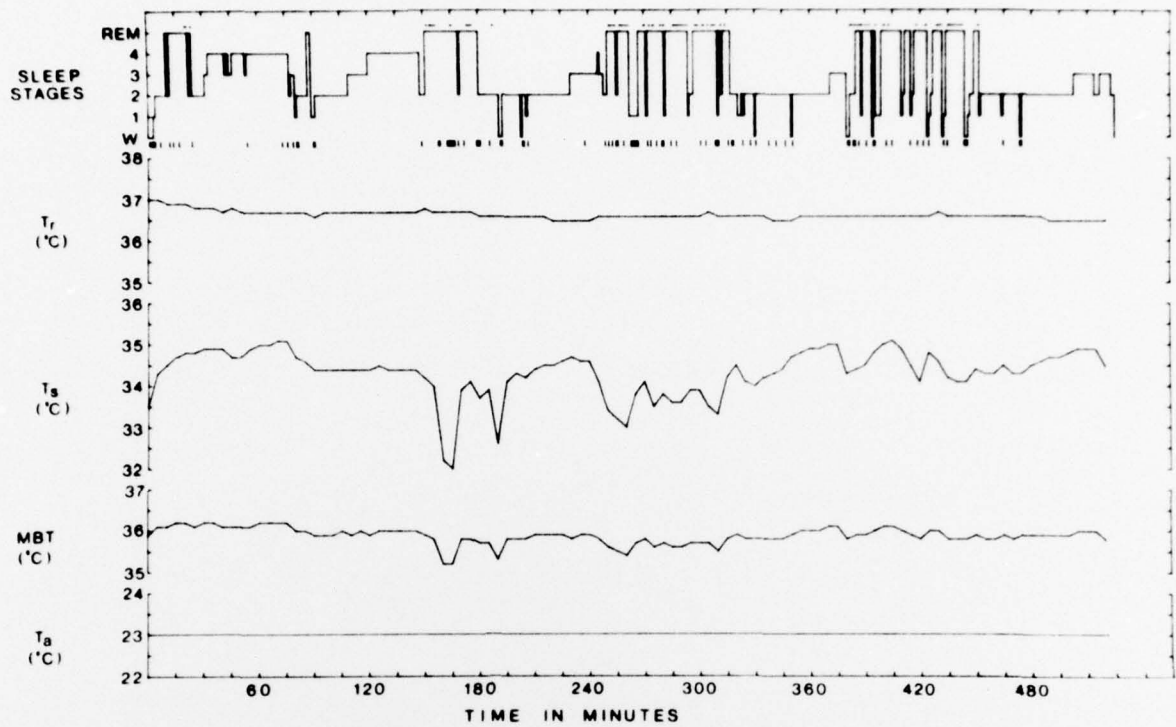
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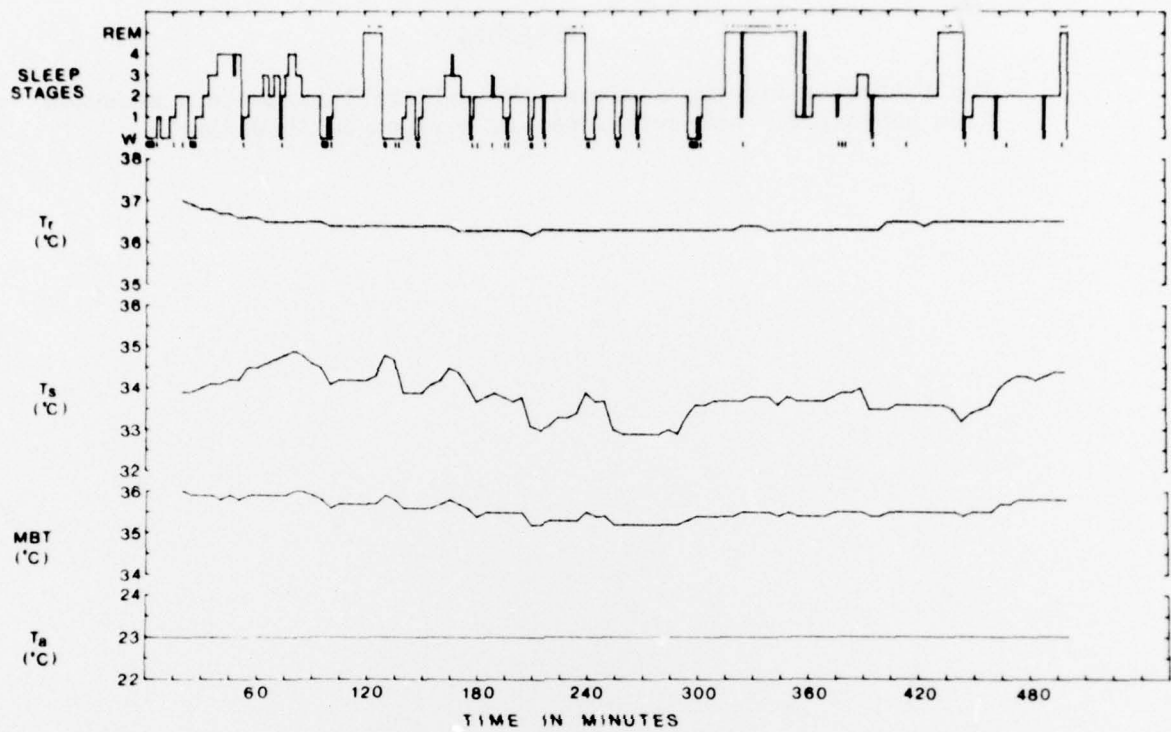


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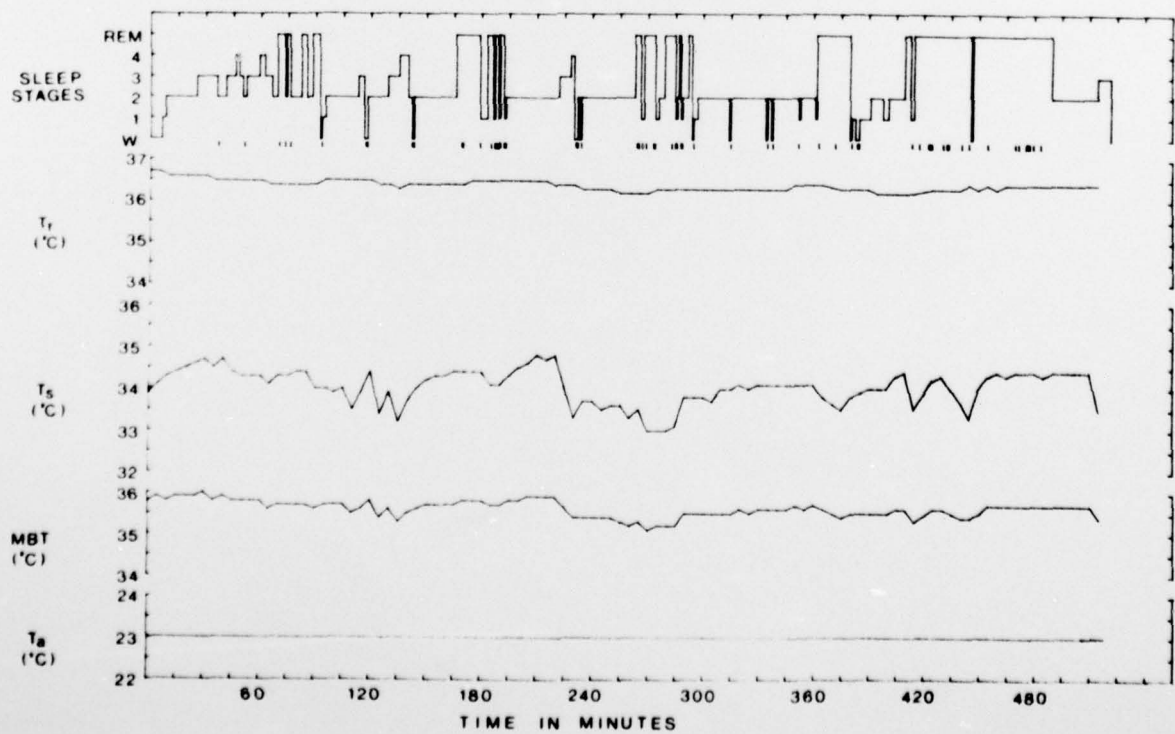
The following figures indicate the different parameters measured from subject 2. Abbreviations are defined in Appendix 1.

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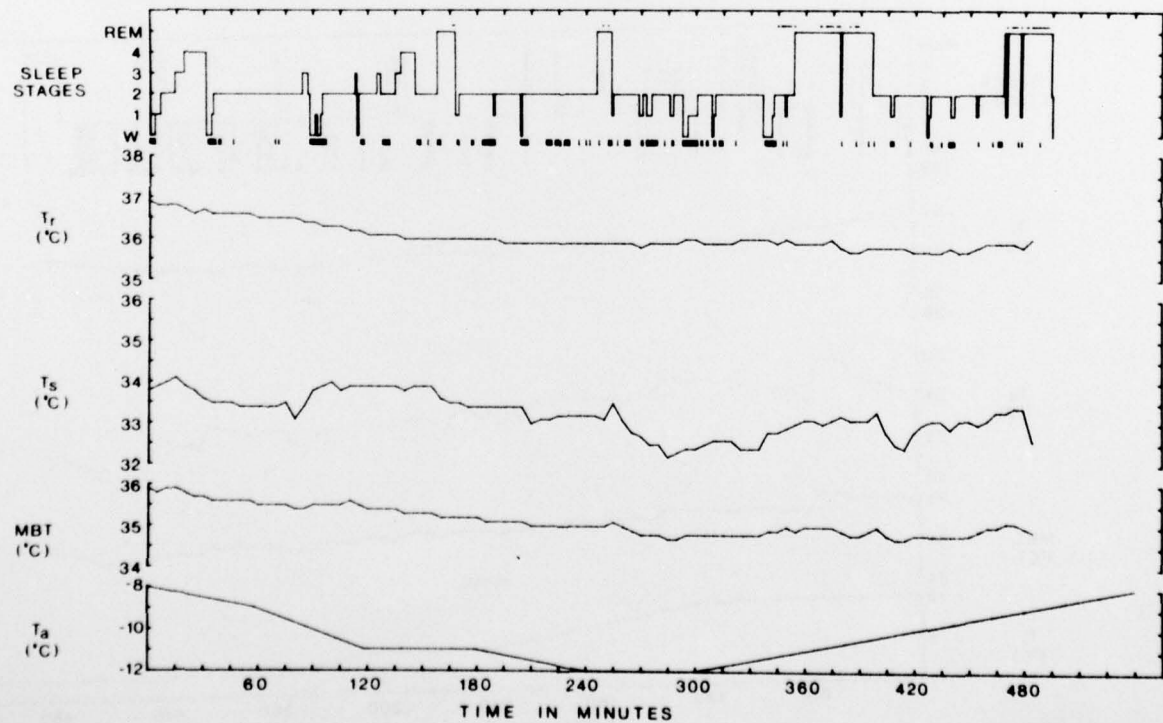
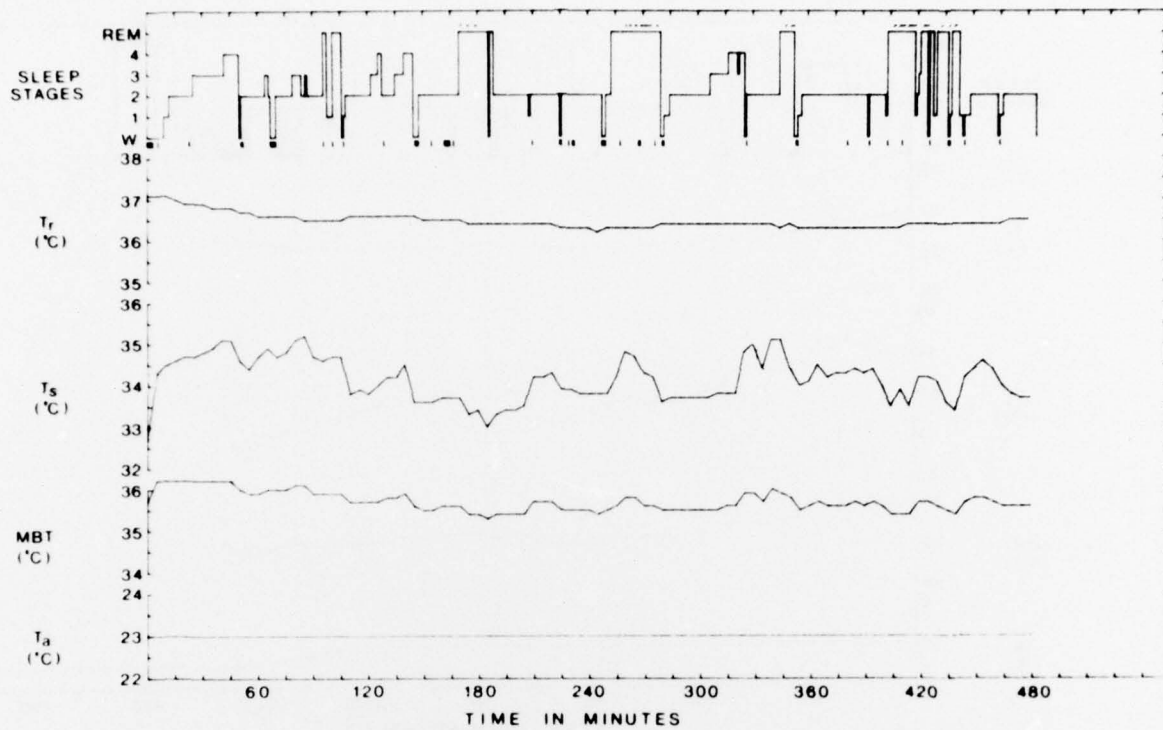
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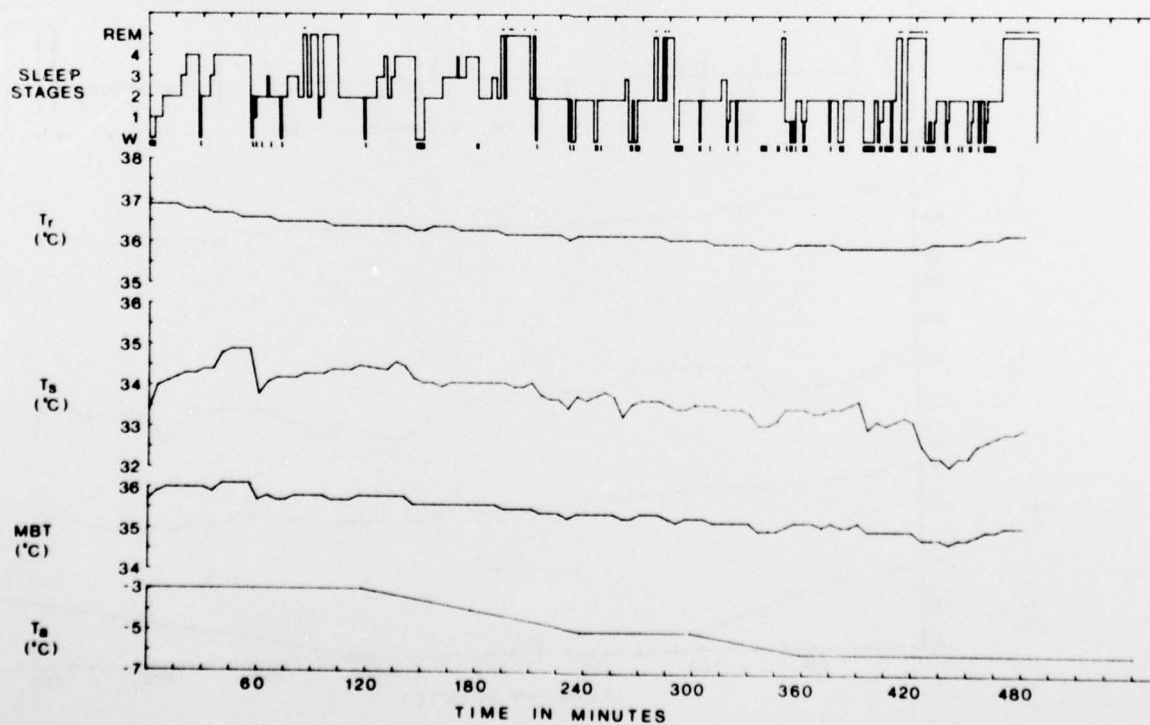
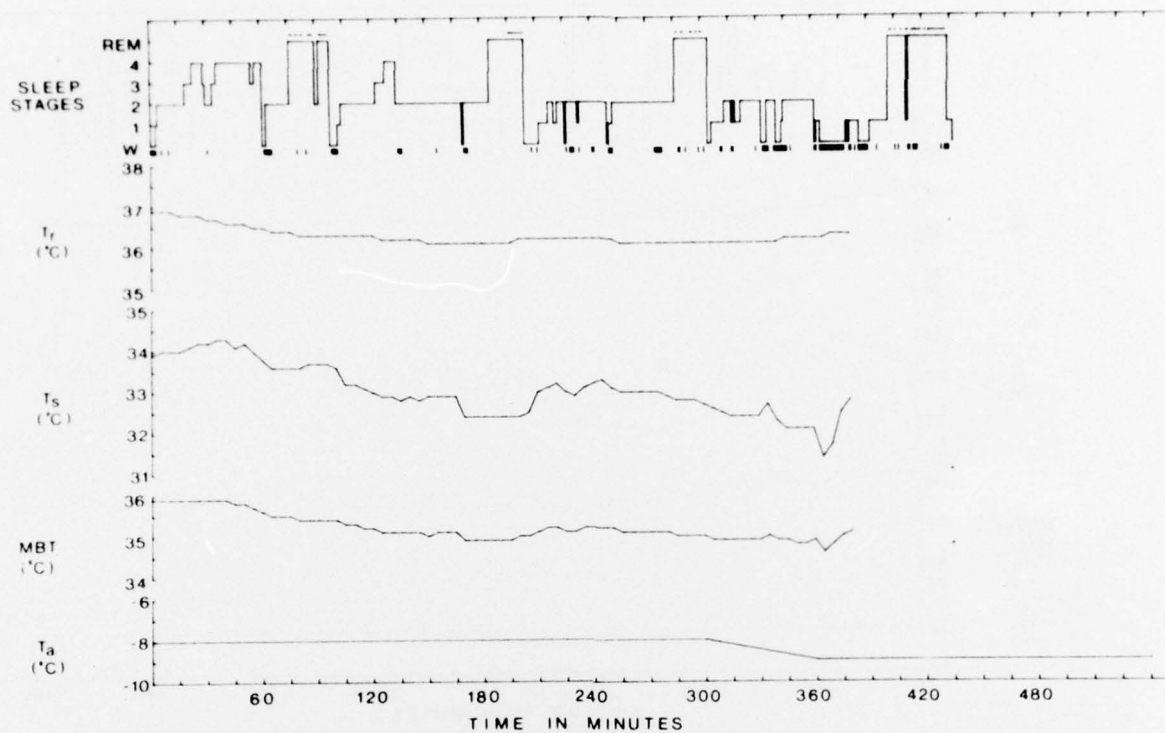


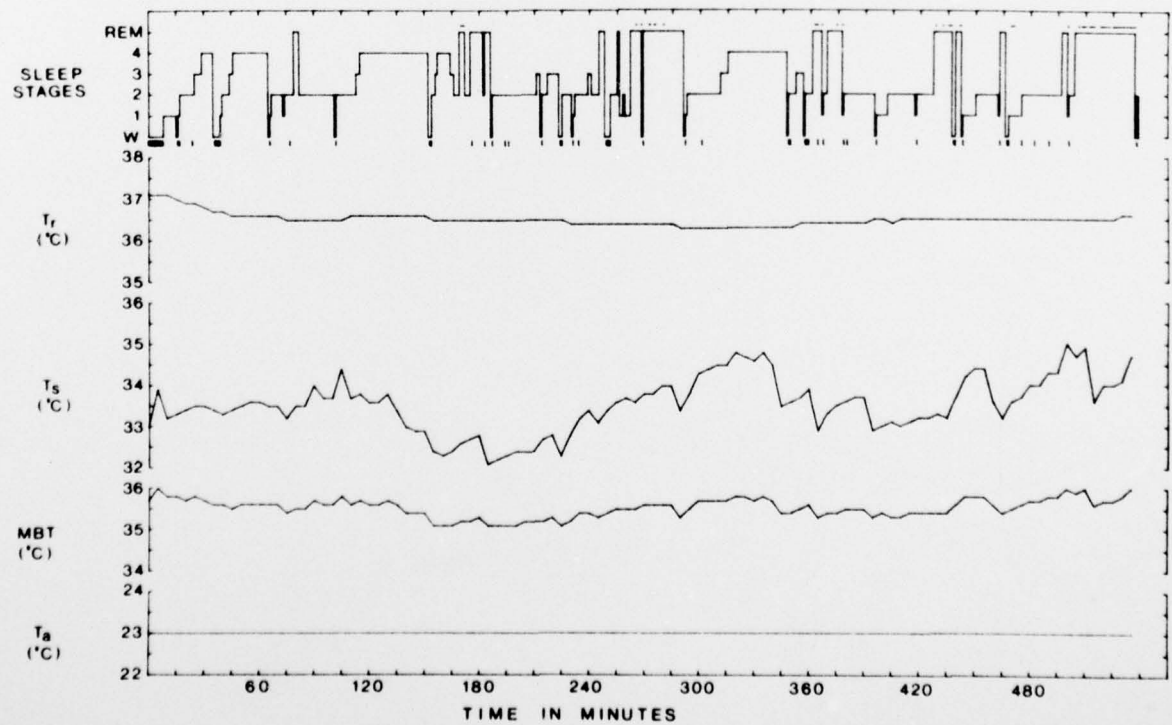
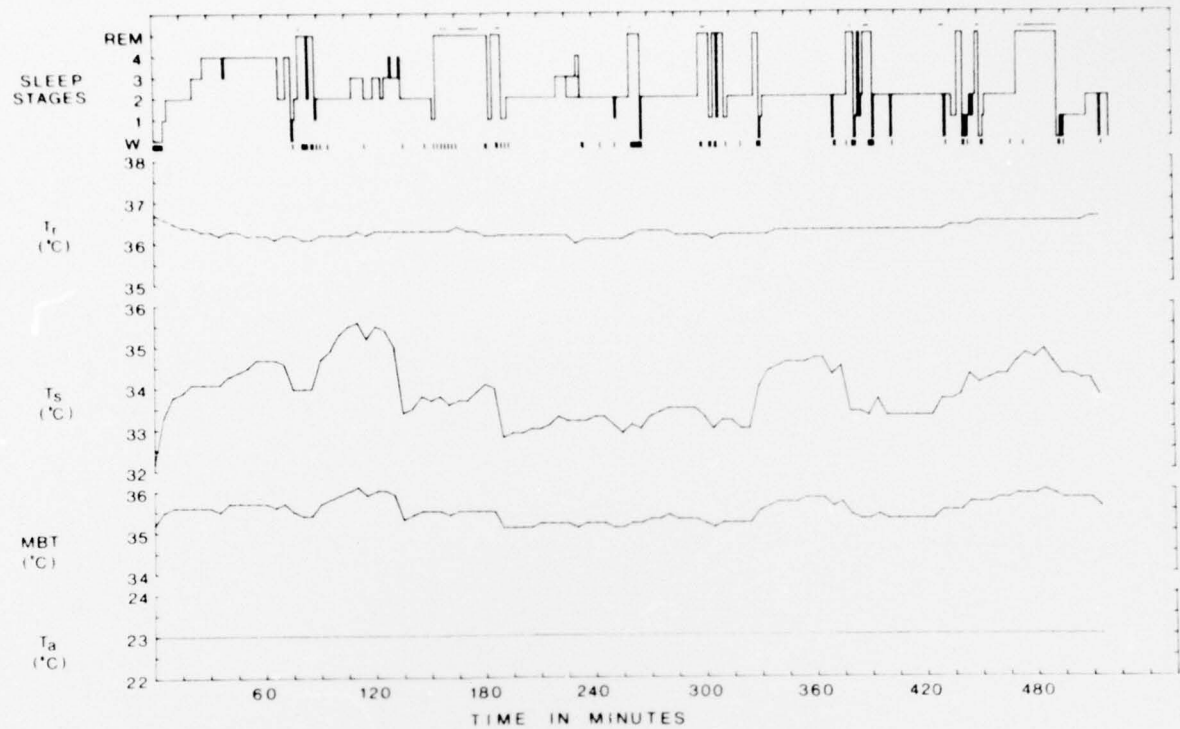
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